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CASE REPORT

Significant Traumatic Intracranial Hemorrhage in the Setting of Massive Bee Venom–Induced Coagulopathy: A Case Report

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Bees and wasps of the Hymenoptera order are encountered on a daily basis throughout the world. Some encounters prove harmless, while others can have significant morbidity and mortality. Hymenoptera venom is thought to contain an enzyme that can cleave phospholipids and cause significant coagulation abnormalities. This toxin and others can lead to reactions ranging from local inflammation to anaphylaxis. We report a single case of a previously healthy man who presented to the emergency department with altered mental status and anaphylaxis after a massive honeybee envenomation that caused a fall from standing resulting in significant head injury. He was found to have significant coagulopathy and subdural bleeding that progressed to near brain herniation requiring emergent decompression. Trauma can easily occur to individuals escaping swarms of hymenoptera. Closer attention must be paid to potential bleeding sources in these patients and in patients with massive bee envenomation.

Key words: hymenoptera, coagulopathy, anaphylaxis, traumatic subdural hemorrhage

Introduction

High venom loads, known as "massive envenomations," result from >50 stings to a victim during a single encounter. This can produce what is known as a toxic reaction, a potentially life-threatening complication that induces anything from rhabdomyolysis to disseminated intravascular coagulation. We present a case of a patient who experienced significant intracranial bleeding and complications immediately following massive bee envenomation, which produced coagulopathy after head trauma.

Case Presentation

A 71-year-old man with no prior medical history for significant disease presented to the emergency department (ED) with altered mental status, a reported fall, and anaphylaxis. The patient was a groundskeeper at a local high school where he was using a weed-whacker in a level grass field when he disturbed a beehive. His coworkers reported they found him on the ground where he had either tripped and fallen from standing or syncopized; he was covered in honeybees to the extent that bystanders were unable to see his face or neck. The school nurse administered intramuscular epinephrine 0.3 mg prior to emergency medical services (EMS) arrival as he appeared unresponsive to bystanders.

Upon EMS arrival, the patient was combative, with altered mental status, and found to be in acute anaphylaxis with reported vital sign abnormalities including a blood pressure of 96/70 mm Hg, heart rate of 120 beats/min, and respiratory rate of 20 breaths/min with wheezing. His initial Glasgow Coma Scale was 10 (eye−4, verbal−1, motor−6) but improved within 5 minutes to a GCS of 14 (eye−4, verbal−4, motor−6). These findings prompted EMS to give a second dose of intramuscular 0.3 mg epinephrine, intravenous (IV) methylprednisolone 125 mg, and 1 L normal saline bolus in the field. Diphenhydramine was not given in the prehospital setting.

In the ED, the patient was agitated, pulling at his cervical collar, and disoriented to place and time. Initial

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vital signs were blood pressure 131/75 mm Hg, heart rate 98 beats/min, respiration rate 25 breaths/min, and oxygen saturation 96% on facemask. On physical examination, a laceration was noted to the back of his head with controlled bleeding and scalp bogginess with equal but sluggishly reactive pupils. Faint wheezes were auscultated throughout all lung fields. He was initially treated with diphenhydramine 50 mg IV, ranitidine 50 mg IV, albuterol nebulizer, and 1 L normal saline fluids for the allergic reaction, and was administered lorazepam 1 mg IV for agitation.

A computed tomography (CT) scan of the head was done within 1 hour of arrival and revealed a right occipital skull facture with associated small left frontal subdural hemorrhage (see Figure 1). Coagulation studies were abnormal with partial thromboplastin time (PTT) >180 seconds, prothrombin time (PT) 21.3 seconds, and international normalized ratio (INR) of 1.7. The initial etiology of these altered bleeding times was unclear at the time as the patient had no known bleeding disorders and took no anticoagulants per the patient's spouse, who was present to give the patient's medical history. A discussion ensued between the emergency medicine physicians and neurosurgical team regarding a possible laboratory abnormality or an altered blood draw while the patient was initially being stabilized, since normal saline was running through the IV simultaneously. The PT, PTT, and INR were all reordered, but due to high patient volume and numerous intensive care unit holds in the ED, these diagnostic studies were delayed and not sent in a timely manner. Hemoglobin and hematocrit were 13.6 g/dL and 40.2%, respectively, and platelet count was $234 \times 10^3/\mu L$. The patient had no signs of renal impairment with a blood urea nitrogen of 17 mg/dL and creatinine of 1.0 mg/dL. Given the small intracranial hemorrhage no operative management was indicated and no coagulation reversal was advised despite acknowledgment of abnormal laboratory results. Supportive care and a repeat CT of the head in 6 hours were recommended. At that time, the patient became much more cooperative and alert and was answering questions more appropriately.

Due to lack of intensive care unit bed availability, the patient remained in the ED 5 hours after arrival. He was continually reassessed, and the nursing staff noted a decline in mental status at the 5.5 hour mark that included moaning and making nonpurposeful movements. The decision was made to repeat a CT scan emergently. Before the scan, he had equal pupillary response. Repeat imaging showed significant increase in the subdural hemorrhage with midline shift and brain compression (see Figure 2). Rapid sequence intubation was performed using etomidate 20 mg and succinylcholine 100 mg with successful intubation, and prothrombin complex concentrate (KCentra, CSL Behring) 2500 units IV and mannitol 25% 100 g was administered in the ED. The hematology service was consulted for the coagulation abnormalities. They surmised that this patient's altered bleeding times were secondary to his significant amount of bee venom exposure. They also postulated these abnormal bleeding times contributed to his traumatic subdural hemorrhage. When the repeat coagulation values did result, they

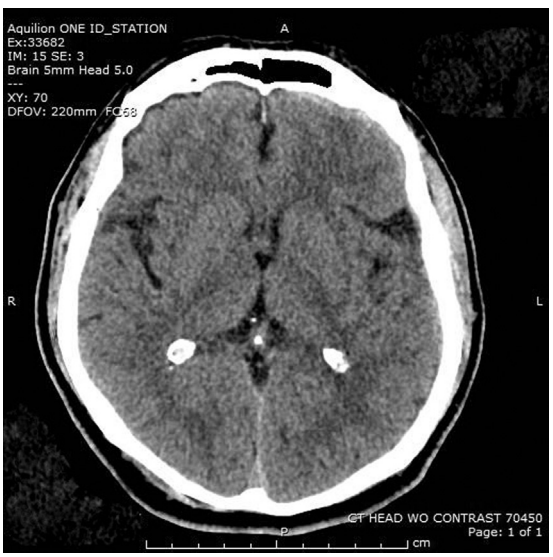

Figure 1. The initial computed tomography scan of our patient, done within the first hour after bee attack, showing a small left frontal subdural hemorrhage.

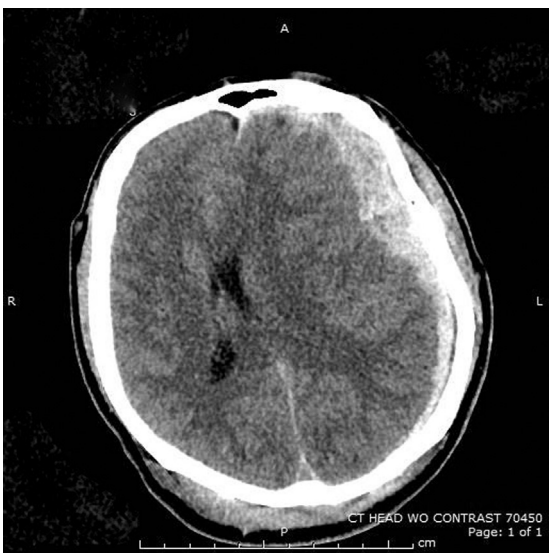

Figure 2. The patient's second computed tomography scan, done about 5.5 hours later, showing significant increase in subdural hemorrhage with midline shift and mass effect.